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PAROXYSMAL VISUAL PHENOMENON, DIAGNOSTIC DILEMMA AND THERAPEUTIC CHALLENGE

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INTRODUCTION

Visual illusions and hallucinations are amongst the most vivid and bizarre symptoms associated with many non-morbid conditions, neurological disorders (such as migraine and epilepsy), psychiatric disorders including schizophrenia, and ophthalmological conditions such as cataracts (Liu et al. 2019, Barodawala & Mulley 1997). The intermittent nature of such experiences and behavioural outbursts can pose diagnostic dilemmas. We report a case of paroxysmal presentation of a visual phenomenon that did not improve with antipsychotics but had a dramatic response to an antiepileptic therapeutic trial.

CASE REPORT

A 20-years-old male presented with intermittent symptoms of visualising intrusive, multi-coloured geometrical designs of triangles, circles, and horizontal lines in various sizes in front of his eyes and altered colour perceptions, induced by flashing lights lasting for 50-60 seconds for six years. He also reported staring spells with screeching sounds, fluttering eye movements, and shivering in the whole body during the episodes with brief post episodic heaviness and a complete recollection post-episode. These would be associated with anger outbursts and self-injurious behaviour. He had a history of birth asphyxia and global developmental delay with a slow to warm premorbid temperament. There was a family history of mood disorder in his paternal grandmother. Before presenting to us, he was diagnosed with chronic psychosis and was on a combination of typical and atypical antipsychotics. He received risperidone (up to four mg/day) for one year, amisulpride (up to 400 mg/day) with lurasidone (up to 40 mg/day) for three months, and thioridazine up to 50 mg/day for two months. His situation gradually worsened despite these medications. Physical examination was unremarkable, while mental status examination suggested simple visual hallucinations, fleeting elementary auditory hallucinations, and dyschromatopsia with impaired judgement and partial insight.

We provisionally diagnosed it as organic hallucinosis with intellectual disability disorder. IQ assessment with WAIS-IV had a score of 73, indicating borderline intelligence. Ophthalmology evaluation was unremarkable. Interictal EEG and MRI brain were normal. However, given the episodic nature of the visual phenomenon, sudden onset, and gradual offset, with a stereotypical pattern of symptoms in each episode associated with behavioural outrage and post-event heaviness, we considered the possibility of an ictal event (focal sensory seizures, simple partial to complex partial) more likely, than a diagnosis of brief intermittent psychosis. We gave a therapeutic trial of carbamazepine that was gradually increased to a dose of 14 mg/kg body weight. Antipsychotics were tapered and stopped, and tablet aripiprazole 10 mg/day was added. He was almost symptom-free by the end of three months. Aripiprazole was tapered and stopped on subsequent visits with no exacerbation of symptoms.

DISCUSSIONS

Epileptic seizures can be mistaken for psychiatric disorders like schizophrenia, brief psychotic disorder, panic disorder, and even Alzheimer's dementia. In our case, a pre-existing psychiatric illness in the background posed a challenge in the management. The patient's description of his visual phenomenon often provides a clue (Liu et al. 2019). However, the episodic symptoms may be difficult to diagnose if they co-occur with behavioural outbursts and can lead to mistaken diagnoses such as brief intermittent psychosis leading to unnecessary antipsychotic exposure. Understanding the clinical presentation is thus of utmost importance (Table 1). As substantial chronic psychosis could not be elicited, and the psychotic exacerbation was paroxysmal without any typical tardive dyskinetic symptoms, the possibility of the dopamine supersensitivity psychosis was ruled out (Fallon & Dursun 2010). Visual aura can be a manifestation of focal epilepsy arising from occipital or temporal regions of the brain without spreading or generalising beyond the interhemispheric commissural pathways. Epilepsy originating from the occipital lobe

Table 1. Differential diagnosis for paroxysmal visual phenomenon with behavioural abnormalities

Differentiating points	Epilepsy	Brief Intermittent Psychosis	Migraine
Preceding symptoms	Non-specific prodromal symptoms	Acute stress reaction in some cases	Non-specific prodromal symptoms
Visual phenomenon	Occipital origin: optical illusions (metamorphopsia, dyschromatopsia), ill-formed visual hallucinations, ictal blindness Medial Temporal origin: well-formed visual hallucinations	Usually rare, If present may indicate effect of substance abuse or organic etiology	Visual illusions/ hallucinations (stars, flashes, or simple geometric shapes); enlarging scintillating scotomas, fortification spectra; transient amaurosis, hemianopias, cortical blindness
Associated symptoms	Intact/lack of consciousness, automatisms Occipital origin: Eye and head deviation, eyelid fluttering Medial Temporal origin: autonomic, affective and behavioural symptoms, De-Ja Vu phenomena	Intact consciousness Pan-anxiety, fleeting or well-formed delusions, hallucinations, formal thought disorders, unprovoked agitation	Intact consciousness Pulsatile headache associated with photophobia, phonophobia, nausea or vomiting
Post episode symptoms	Post-ictal headache, lethargy, confusion, nausea and vomiting	Residual psychosis or affective symptoms	-
Duration of episode	Few seconds to minutes	Few hours to days	Few hours
Investigations	EEG can aid diagnosis; Brain imaging can identify possible structural pathology	Diagnostic psychometry can be useful	Clinical diagnosis
Treatment	Responds to antiepileptics, May worsen with antipsychotics	Responds well to antipsychotics, Poor evidences for antiepileptics	Responds to non-steroidal analgesics, and antimigraine medications
Course and prognosis	Episodic, Usually, well-controlled with antiepileptics	Can progress to schizophrenia	Episodic, Usually, well-controlled with prophylactic antimigraine medications

presents with elementary visual hallucinations involving the primary visual cortex or associated fields with other typical features of optical illusions like metamorphopsia or dyschromatopsia, ictal blindness, eye and head deviation, eyelid fluttering, and postictal headache (Adcock & Panayiotopoulos 2012). Anteromedial temporal lobe epilepsy could also present with similar features and autonomic, affective, and behavioural symptoms owing to the involvement of the temporal-limbic structures (Bien et al. 2000). The onset is at a much younger age in occipital lobe epilepsy (Adcock & Panayiotopoulos 2012, Beletsky & Mirsattari 2012). Thus, intermittent organic visual hallucinations with onset at an older age were possibly a manifestation of epilepsy originating from the anteromedial temporal cortex spreading to the medial temporal region resulting in simple visual hallucinations, dyschromatopsia, autonomic symptoms, and associated behavioural outbursts. The surface EEG is not always helpful in such cases (Smith 2005). Also, the therapeutic trial of carbamazepine leading to complete control of the episodes further substantiates the ictal aetiology.

CONCLUSIONS

The presence of paroxysmal sensory distortion and deception associated with behavioural outbursts may mimic psychosis; however, nonresponse to antipsychotics, with an abrupt onset and offset, and stereotypic episodes should always raise the possibility of an ictal event. A therapeutic trial of antiepileptic drugs may be indicated if there is a strong clinical suspicion even in the absence of corroborative EEG or neuroimaging findings (Coerver & Subramanian 2020, Weis et al. 2005).

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Declaration of patient consent*:*

The authors certify that they have obtained an appropriate patient consent form. In the form, the patient has given his/her consent for his/her clinical information to be reported in the journal without disclosure of his/her identity.

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Sharon Narula: data collection and first draft.

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